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NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	JAN 12	Match STN Content and Features to Your Information Needs, Quickly and Conveniently
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NEWS 4	FEB 16	STN Express Maintenance Release, Version 8.4.2, Is Now Available for Download
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NEWS 6	FEB 16	New FASTA Display Formats Added to USGENE and PCTGEN
NEWS 7	FEB 16	INPADOCDB and INPAFAMDB Enriched with New Content and Features
NEWS 8	FEB 16	INSPEC Adding Its Own IPC codes and Author's E-mail Addresses
NEWS 9	APR 02	CAS Registry Number Crossover Limits Increased to 500,000 in Key STN Databases
NEWS 10	APR 02	PATDPAFULL: Application and priority number formats enhanced
NEWS 11	APR 02	DWPI: New display format ALLSTR available
NEWS 12	APR 02	New Thesaurus Added to Derwent Databases for Smooth Sailing through U.S. Patent Codes
NEWS 13	APR 02	EMBASE Adds Unique Records from MEDLINE, Expanding Coverage back to 1948
NEWS 14	APR 07	CA/CAplus CLASS Display Streamlined with Removal of Pre-IPC 8 Data Fields
NEWS 15	APR 07	50,000 World Traditional Medicine (WTM) Patents Now Available in CAplus
NEWS 16	APR 07	MEDLINE Coverage Is Extended Back to 1947

NEWS EXPRESS FEBRUARY 15 10 CURRENT WINDOWS VERSION IS V8.4.2,
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* * * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * * *

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FILE 'HOME' ENTERED AT 11:19:38 ON 10 MAY 2010

=> file caplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.22       0.22

FILE 'CAPLUS' ENTERED AT 11:19:51 ON 10 MAY 2010
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FILE COVERS 1907 - 10 May 2010  VOL 152 ISS 20
FILE LAST UPDATED: 9 May 2010  (20100509/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Feb 2010
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Feb 2010

CAplus now includes complete International Patent Classification (IPC)
reclassification data for the first quarter of 2010.

CAS Information Use Policies apply and are available at:

http://www.cas.org/legal/infopolicy.html

This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> s disulfiram
          2293 DISULFIRAM
             2 DISULFIRAMS
          2293 DISULFIRAM
                (DISULFIRAM OR DISULFIRAMS)
            10 DISULPHIRAM
L1        2300 DISULFIRAM
                (DISULFIRAM OR DISULPHIRAM)

=> s disulfiram/cn
   REGISTRY INITIATED
Substance data SEARCH and crossover from CAS REGISTRY in progress...
Use DISPLAY HITSTR (or FHITSTR) to directly view retrieved structures.




L3        3380 L2

=> s disulfram/cn
   REGISTRY INITIATED
```

```
Substance data SEARCH and crossover from CAS REGISTRY in progress...
Use DISPLAY HITSTR (or FHITSTR) to directly view retrieved structures.



L5            0 L4

=> s curcumin
          6749 CURCUMIN
            75 CURCUMINS
L6        6756 CURCUMIN
                (CURCUMIN OR CURCUMINS)

=> file registry
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                        ENTRY    SESSION
FULL ESTIMATED COST                                      2.81      18.32

FILE 'REGISTRY' ENTERED AT 11:20:42 ON 10 MAY 2010
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Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES:    9 MAY 2010  HIGHEST RN 1221824-45-8
DICTIONARY FILE UPDATES:   9 MAY 2010  HIGHEST RN 1221824-45-8

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TSCA INFORMATION NOW CURRENT THROUGH January 8, 2010.

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

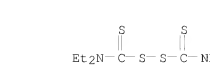
http://www.cas.org/support/stngen/stndoc/properties.html

=> s disulfiram/cn
L7            1 DISULFIRAM/CN

=> d l7

L7   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2010 ACS on STN
RN   97-77-8  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Thioperoxydicarbonic diamide ([(H2N)C(S)]2S2), N,N,N',N'-tetraethyl-  (CA
     INDEX NAME)
OTHER CA INDEX NAMES:
CN   Disulfide, bis(diethylthiocarbamoyl) (8CI)
OTHER NAMES:
CN   Abstensil
CN   Abstinil
```

CN Abstinyl
CN Accel TET
CN Accel TET-R
CN Akrochem TETD
CN Alcophobin
CN Antabus
CN Antabuse
CN Antadix
CN Antaethyl
CN Antalcol
CN Antetan
CN Antetil
CN Anticol
CN Antietanol
CN Antietil
CN Antikol
CN Antivitium
CN Aversan
CN Averzan
CN Bis(diethylthiocarbamoyl) disulfide
CN Bis(N,N-diethylthiocarbamoyl) disulfide
CN Contralin
CN Cronetal
CN Dicupral
CN Disulfiram
CN Ekagom DTET
CN Ekagom TEDS
CN Ekagom TETDS
CN Espenal
CN Esperal
CN Etabus
CN Ethyl Thiram
CN Ethyl Thiurad
CN Ethyl Tuads
CN Ethyl Tuex
CN Etiltox
CN Exhoran
CN Exhorran
CN Hoca
CN Krotenal
CN N,N,N',N'-Tetraethylthiuram disulfide
CN Nocceler TED
CN Nocceler TET
CN Nocceler TET-G
CN Noxal
CN NSC 25953
CN Refusal
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
DR 11078-22-1, 155-01-1
MF C10 H20 N2 S4
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DRUGU, EMBASE, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IMSDRUGNEWS, IMSPRODUCT, IMSRESEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS, PROMT, PS, RTECS*, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL, USPATOLD
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3369 REFERENCES IN FILE CA (1907 TO DATE)
72 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
3380 REFERENCES IN FILE CAPLUS (1907 TO DATE)

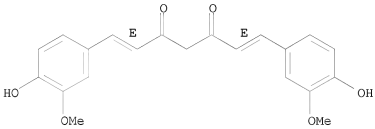
=> s curcumin/cn
L8 1 CURCUMIN/CN

=> d 18

L8 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2010 ACS on STN
RN 458-37-7 REGISTRY
ED Entered STN: 16 Nov 1984
CN 1,6-Heptadiene-3,5-dione, 1,7-bis(4-hydroxy-3-methoxyphenyl)-, (1E,6E)-
(CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 1,6-Heptadiene-3,5-dione, 1,7-bis(4-hydroxy-3-methoxyphenyl)-, (E,E)-
(8CI)
CN Curcumin (6CI)
OTHER NAMES:
CN (1E,6E)-1,7-Bis(4-hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione
CN (E)-1,7-Bis(4-hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione
CN (E,E)-1,7-Bis(4-hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione
CN C Yellow 15
CN C.I. 75300
CN C.I. Natural Yellow 3
CN Curcuma
CN Curcumin I
CN Curcumine
CN Diferuloylmethane
CN E 100
CN E 100 (dye)
CN Haidr
CN Halad
CN Haldar
CN Halud
CN Indian Saffron
CN Jianghuangsu
CN Kacha Haldi
CN Merita Earth
CN Natural Yellow 3
CN NSC 32982
CN San-Ei Curcumine AL
CN San-Ei Gen Curcumine AL
CN Souchet
CN Terra Merita
CN trans,trans-Curcumin
CN Turmeric
CN Turmeric (dye)
CN Turmeric yellow
CN Ukon
CN Ukon (dye)
CN Yellow Ginger

CN Yellow Root
CN Yo-Kin
FS STEREOSEARCH
DR 15845-47-3, 73729-23-4, 79257-48-0, 91884-86-5, 33171-04-9
MF C21 H20 O6
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, IMSPRODUCT, IMSRESEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, PIRA, PROMT, PROUSDDR, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, ULIDAT, USPAT2, USPATFULL, USPATOLD
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Double bond geometry as shown.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

5369 REFERENCES IN FILE CA (1907 TO DATE)
237 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
5428 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> s BSO/cn
L9 1 BSO/CN

=> d l9

L9 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2010 ACS on STN
RN 12377-72-9 REGISTRY
ED Entered STN: 16 Nov 1984
CN Bismuth oxide silicate (Bi12O16(SiO4)) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Bismuth silicate (Bi12SiO20) (7CI)
OTHER NAMES:
CN Bismuth oxide silicate
CN Bismuth oxide silicate (Bi12SiO20)
CN Bismuth silicon oxide (6Bi2O3.SiO2)
CN Bismuth silicon oxide (Bi12SiO20)
CN BSO
CN Silicosillenite
DR 849060-15-7, 66256-73-3, 225239-83-8, 398473-14-8
MF Bi . O4 Si . O
AF Bi12 O20 Si
CI TIS
LC STN Files: CA, CAPLUS, CHEMLIST, IFICDB, IFIPAT, IFIUDB, TOXCENTER, USPAT2, USPATFULL
Other Sources: EINECS**, TSCA**

(**Enter CHEMLIST File for up-to-date regulatory information)

Component	Ratio	Component Registry Number
O	16	17778-80-2
O4Si	1	17181-37-2
Bi	12	7440-69-9

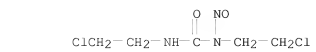
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1947 REFERENCES IN FILE CA (1907 TO DATE)
24 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1947 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> s BCNU/cn
L10 1 BCNU/CN

=> d l10

L10 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2010 ACS on STN
RN 154-93-8 REGISTRY
ED Entered STN: 16 Nov 1984
CN Urea, N,N'-bis(2-chloroethyl)-N-nitroso- (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Urea, 1,3-bis(2-chloroethyl)-1-nitroso- (8CI)
OTHER NAMES:
CN 1,3-Bis(β-chloroethyl)-1-nitrosourea
CN 1,3-Bis(2-chlorethyl)-1-nitrosourea
CN 1,3-Bis(2-chloroethyl)-1-nitrosourea
CN BCNU
CN Becenun
CN BiCNU
CN Carmubris
CN Carmustin
CN Carmustine
CN DTI 015
CN FDA 0345
CN Gliadel
CN Gliadel Wafer
CN N,N'-Bis(2-chloroethyl)-N-nitrosourea
CN Nitrumon
CN NSC 409962
CN SK 27702
CN SRI 1720
DR 1159711-15-5, 1191292-23-5
MF C5 H9 Cl2 N3 O2
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DRUGU, EMBASE, IPA, MEDLINE, MRCK*, MSDS-OHS, PATDPASPC, PROMT, PROUSDDR, PS, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3831 REFERENCES IN FILE CA (1907 TO DATE)
79 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
3851 REFERENCES IN FILE CAPLUS (1907 TO DATE)

```
=> d his

     (FILE 'HOME' ENTERED AT 11:19:38 ON 10 MAY 2010)

     FILE 'CAPLUS' ENTERED AT 11:19:51 ON 10 MAY 2010
L1         2300 S DISULFIRAM
                S DISULFIRAM/CN

     FILE 'REGISTRY' ENTERED AT 11:20:06 ON 10 MAY 2010
L2            1 S DISULFIRAM/CN

     FILE 'CAPLUS' ENTERED AT 11:20:06 ON 10 MAY 2010
L3         3380 S L2
                S DISULFRAM/CN

     FILE 'REGISTRY' ENTERED AT 11:20:23 ON 10 MAY 2010
L4            0 S DISULFRAM/CN

     FILE 'CAPLUS' ENTERED AT 11:20:23 ON 10 MAY 2010
L5            0 S L4
L6         6756 S CURCUMIN

     FILE 'REGISTRY' ENTERED AT 11:20:42 ON 10 MAY 2010
L7            1 S DISULFIRAM/CN
L8            1 S CURCUMIN/CN
L9            1 S BSO/CN
L10           1 S BCNU/CN

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COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY     SESSION
FULL ESTIMATED COST                                30.89       49.21

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FILE COVERS 1907 - 10 May 2010  VOL 152 ISS 20
FILE LAST UPDATED: 9 May 2010  (20100509/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Feb 2010
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Feb 2010

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reclassification data for the first quarter of 2010.

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This file contains CAS Registry Numbers for easy and accurate
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=> s l7
L11       3380 L7

=> s l8
L12       5428 L8

=> s l9
L13       1947 L9

=> s l10
L14       3851 L10

=> s cancer or tumor or neoplasm
         454671 CANCER
          66711 CANCERS
         471046 CANCER
                (CANCER OR CANCERS)
         543550 TUMOR
         195321 TUMORS
         602918 TUMOR
                (TUMOR OR TUMORS)
           4848 TUMOUR
           1830 TUMOURS
           6560 TUMOUR
                (TUMOUR OR TUMOURS)
         603365 TUMOR
                (TUMOR OR TUMOUR)
         593577 NEOPLASM
          38826 NEOPLASMS
         610989 NEOPLASM
                (NEOPLASM OR NEOPLASMS)
L15    1004074 CANCER OR TUMOR OR NEOPLASM

=> s l11 and l15
L16        224 L11 AND L15

=> s l12 and l15
L17       1654 L12 AND L15

=> s l13 and l15
L18          3 L13 AND L15

=> s l14 and l15
L19       2789 L14 AND L15

=> s (l16 or l17) and (l18 or l19)
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L20         34 (L16 OR L17) AND (L18 OR L19)

=> dup rem l20
PROCESSING COMPLETED FOR L20
L21          34 DUP REM L20 (0 DUPLICATES REMOVED)

=> s l21 and ad<20030718
L22         34 S L21
       4690081 AD<20030718
                (AD<20030718)
L23         10 L22 AND AD<20030718

=> d l23 1-10 ibib abs

L23  ANSWER 1 OF 10  CAPLUS  COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER:         2007:507420  CAPLUS
DOCUMENT NUMBER:          146:475663
TITLE:                    Compositions and methods for the treatment of
                          cancer
INVENTOR(S):              D'Andrea, Alan D.; Taniguchi, Toshiyasu
PATENT ASSIGNEE(S):       Dana Farber Cancer Institute, USA
SOURCE:                   U.S. Pat. Appl. Publ., 49pp., Cont.-in-part of U.S.
                          Ser. No. 46,346.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   5
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.          DATE
     ----------------    ----   --------    --------------------     --------
     US 20070105130       A1    20070510    US 2006-441289           20060524
     US 20030093819       A1    20030515    US 2001-998027           20011102 <--
     US 20030188326       A1    20031002    US 2002-165099           20020606 <--
     US 20050255502       A1    20051117    US 2005-46346            20050128
     US 7459287           B2    20081202
     US 20090186355       A1    20090723    US 2008-315368           20081201
PRIORITY APPLN. INFO.:                       US 2000-245756P      P   20001103
                                             US 2001-998027       B2  20011102
                                             US 2002-165099       A2  20020606
                                             US 2004-540380P      P   20040130
                                             US 2005-46346        A2  20050128
                                             US 2005-684136P      P   20050524
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB   Disclosed herein are methods and compns. for the treatment of
     cancer.  In particular, the present invention discloses inhibitors
     of the Fanconi anemia pathway and methods using same.  Such inhibitors are
     useful in inhibiting DNA damage repair and can be useful, for example, in
     the treatment of cancer.  These agents can be combined with
     genotoxic antineoplastic agents.  In one aspect, the invention provides a
     method of predicting whether a subject with a neoplastic disorder or
     disease will respond to a genotoxic antineoplastic agent.  The method
     comprises obtaining a biol. sample from the subject, and determining degree of
     ubiquitination of the Fanconi anemia complementation group D2 (FANC D2)
     polypeptide within the biol. sample.  In another aspect, a method of
     identifying an inhibitor of a non-Fanconi anemia DNA damage repair pathway
     is provided.  The method comprises the following steps: (a) providing a
     control cell that is functional in the Fanconi anemia pathway; (b)
     providing a test cell that is isogenic to the test cell but is defective
     in the Fanconi anemia pathway; (c) contacting the test cell and the
     control cell with a test compound; and, (d) comparing the sensitivity of the
     test cell and said control cell to the test compound
```

L23 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2007:175576 CAPLUS
DOCUMENT NUMBER: 146:258964
TITLE: Method for augmentation of intraepithelial and systemic exposure of therapeutic agents having substrate activity for cytochrome p450 enzymes and membrane efflux systems following vaginal and oral cavity administration
INVENTOR(S): Pauletti, Giovanni M.; Harrison, Donald C.; Desai, Kishorkumar J.
PATENT ASSIGNEE(S): Histogenics Corp., USA
SOURCE: U.S. Pat. Appl. Publ., 24 pp., Cont.-in-part of U.S. Ser. No. 208,209.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 12
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20070036834	A1	20070215	US 2006-522126	20060915
AU 765269	B2	20030911	AU 2001-54192	20010703 <--
US 20030049302	A1	20030313	US 2002-226667	20020821 <--
US 6982091	B2	20060103		
US 20060002966	A1	20060105	US 2005-208209	20050818
AU 2006292507	A1	20070329	AU 2006-292507	20060915
CA 2622746	A1	20070329	CA 2006-2622746	20060915
WO 2007035515	A2	20070329	WO 2006-US36087	20060915
WO 2007035515	A3	20070927		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
EP 1948103	A2	20080730	EP 2006-824976	20060915
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
JP 2009508869	T	20090305	JP 2008-531372	20060915

PRIORITY APPLN. INFO.:

US 2001-315877P	P	20010829
US 2002-226667	A1	20020821
US 2005-208209	A2	20050818
US 2005-717680P	P	20050915
AU 1998-76976	A3	19980610
WO 2006-US36087	W	20060915

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The present invention relates to a method for augmentation of epithelial concentration and systemic exposure of therapeutic agents having a substrate affinity for cytochrome P 450 enzymes and membrane efflux transporter systems by using a vaginal or buccal drug delivery compns. and/or devices. Specifically, the invention relates to a method for augmentation of intraepithelial concentration and/or systemic bioavailability for delivery of anti-viral and/or anti-cancer therapeutic agents having a substrate affinity for cytochrome P 450 enzymes and membrane efflux

systems by using a vaginal or buccal drug delivery of these drugs into the systemic circulation by delivering such drug to a subject in need thereof vaginally or buccally in an especially formulated composition increasing the drug's bioavailability by providing means for increasing the drug solubility and permeability through the vaginal or buccal mucosa.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)

L23 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2006:606492 CAPLUS
DOCUMENT NUMBER: 145:76623
TITLE: Compounds and methods for thiol-containing compound efflux and cancer treatment
INVENTOR(S): Day, Brian J.; Kachadourian, Remy
PATENT ASSIGNEE(S): National Jewish Medical and Research Center, USA
SOURCE: U.S. Pat. Appl. Publ., 62 pp., Cont.-in-part of U.S. Ser. No. 400,980.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20060135585	A1	20060622	US 2005-280959	20051115
US 20040087527	A1	20040506	US 2003-400980	20030327 <--
AU 2006327105	A1	20070628	AU 2006-327105	20061115
CA 2669503	A1	20070628	CA 2006-2669503	20061115
WO 2007073518	A2	20070628	WO 2006-US60941	20061115
WO 2007073518	A9	20070823		
WO 2007073518	A3	20071025		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

EP 1954681	A2	20080813	EP 2006-848736	20061115

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR

PRIORITY APPLN. INFO.:

US 2002-422802P	P	20021031
US 2003-400980	A2	20030327
US 2005-280959	A	20051115
WO 2006-US60941	W	20061115

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 145:76623
AB Methods for therapy of cystic fibrosis and other conditions such as cancer are provided. The methods comprise one or more agents capable of increasing thiol-containing compound transport via a transporter system (i.e.ABC transporters such as MDR-1 or MRP-2) in cells. Other embodiments include the use of agents to modulate transport of thiol-containing compds. within the cell. Therapeutic methods involve the administration of such agents to a patient afflicted with cystic fibrosis, cancer and/or another condition responsive to stimulation of

thiol-containing compound transport.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L23 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2004:80356 CAPLUS
DOCUMENT NUMBER: 140:139468
TITLE: Method of inhibiting ATF/CREB and cancer cell growth and pharmaceutical compositions for same
INVENTOR(S): Kennedy, Thomas Preston
PATENT ASSIGNEE(S): Charlotte-Mecklenburg Hospital Authority, USA
SOURCE: U.S. Pat. Appl. Publ., 22 pp., Cont.-in-part of U.S. Ser. No. 392,122.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20040019102	A1	20040129	US 2003-437477	20030514 <--
US 20030065026	A1	20030403	US 1999-392122	19990908 <--
US 6589987	B2	20030708		
CA 2525829	A1	20050203	CA 2004-2525829	20040513
WO 2005009338	A2	20050203	WO 2004-US15283	20040513
WO 2005009338	A3	20050224		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1622599	A2	20060208	EP 2004-776013	20040513

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK

PRIORITY APPLN. INFO.:	US 1998-99390P	P 19980908
	US 1999-392122	A2 19990908
	US 2003-437477	A 20030514
	WO 2004-US15283	W 20040513

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB There is provided a method for inhibiting ATF/CREB and cancer cell growth using disulfiram, administered in combination with heavy metals. It was found that disulfiram disrupts transcription factor DNA binding by forming mixed disulfides with thiols within the DNA-binding region, and that this process is facilitated by metal ions. Disulfiram administered to melanoma cells in combination with copper (II) or zinc(II) decreased expression of cyclin A, reduced proliferation in vitro, and inhibited growth of melanoma cells. The combination of oral zinc gluconate and disulfiram at currently approved doses for alcoholism stabilized tumor growth in two of three patients with Stage IV metastatic melanoma, with 12 and 17 mo survivals, resp., to date, and produced a >50% reduction in hepatic metastases in one individual.
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

L23 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2002:778592 CAPLUS
DOCUMENT NUMBER: 137:259666
TITLE: High-throughput stem cell assay of hematopoietic stem and progenitor cell proliferation
INVENTOR(S): Rich, Ivan N.
PATENT ASSIGNEE(S): Hemogenix, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 29 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 20020146680	A1	20021010	US 2002-59521	20020129	<--
US 7354729	B2	20080408			
CA 2437084	A1	20030116	CA 2002-2437084	20020129	<--
WO 2003004995	A2	20030116	WO 2002-US2458	20020129	<--
WO 2003004995	A3	20030522			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2002335610	A1	20030121	AU 2002-335610	20020129	<--
AU 2002335610	B2	20080417			
EP 1364197	A2	20031126	EP 2002-770372	20020129	<--
EP 1364197	B1	20100414			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
AT 464560	T	20100415	AT 2002-770372	20020129	<--
US 20040110243	A1	20040610	US 2003-645077	20030821	
US 7354730	B2	20080408			
US 20070148668	A1	20070628	US 2006-561133	20061117	
US 7666615	B2	20100223			
US 20080160563	A1	20080703	US 2008-49815	20080317	
US 7700354	B2	20100420			
US 20080160564	A1	20080703	US 2008-49861	20080317	
US 7709258	B2	20100504			
US 20080160544	A1	20080703	US 2008-49921	20080317	
US 20090011446	A1	20090108	US 2008-135021	20080606	

PRIORITY APPLN. INFO.:			
US 2001-264796P	P	20010129	
US 2002-59521	A2	20020129	
WO 2002-US2458	W	20020129	
US 2002-404972P	P	20020821	
US 2003-645077	A2	20030821	
US 2007-942966P	P	20070608	
US 2008-49815	A2	20080317	
US 2008-49921	A2	20080317	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The present invention relates generally to high-throughput assay methods that determine the proliferative status of hematopoietic stem and progenitor cells. The present invention further relates to high-throughput assays for screening compds. that modulate the growth of hematopoietic stem and progenitor cells and for identifying subpopulations thereof that are suitable for transplantation. The assay of the present invention is

particularly useful for quality control and monitoring of the growth potential in the stem cell transplant setting and would provide improved control over the reconstitution phase of transplanted cells.

L23 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2002:555299 CAPLUS
DOCUMENT NUMBER: 137:103875
TITLE: Redox therapy for tumors
INVENTOR(S): Hoffman, Arnold
PATENT ASSIGNEE(S): Israel
SOURCE: PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002056823	A2	20020725	WO 2002-IL51	20020118 <--
WO 2002056823	A3	20071227		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, AP, EA, EP, OA

AU 2002226650	A1	20020730	AU 2002-226650	20020118 <--
US 20040018987	A1	20040129	US 2003-621326	20030718
PRIORITY APPLN. INFO.:			IL 2001-140970 A	20010118
			WO 2002-IL51 W	20020118

AB A method for treating malignancies and/or otherwise controlling the growth and/or proliferative behavior and/or other biol. functions of a cell displaying malignant properties, through the control of the redox state or environment of the cell, preferably through the administration of a GSH-decreasing agent.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L23 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2001:618459 CAPLUS
DOCUMENT NUMBER: 135:190400
TITLE: Method of treating cancer using dithiocarbamate derivatives
INVENTOR(S): Kennedy, Thomas Preston
PATENT ASSIGNEE(S): Charlotte-Mecklenburg Hospital Authority, USA
SOURCE: U.S. Pat. Appl. Publ., 36 pp., Cont.-in-part of U.S. Ser. No. 679,932.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20010016600	A1	20010823	US 2000-735205	20001212 <--
US 6548540	B2	20030415		

US 20030065026	A1	20030403	US 1999-392122	19990908	<--
US 6589987	B2	20030708			
US 6706759	B1	20040316	US 2000-679932	20001005	<--
CA 2424761	A1	20020411	CA 2001-2424761	20011004	<--
WO 2002028349	A2	20020411	WO 2001-US31142	20011004	<--
WO 2002028349	A3	20020711			

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2001096610	A	20020415	AU 2001-96610	20011004	<--
EP 1328267	A2	20030723	EP 2001-977495	20011004	<--
EP 1328267	B1	20081126			

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

JP 2004525079	T	20040819	JP 2002-531975	20011004	<--
JP 4268801	B2	20090527			
AU 2001296610	B2	20060629	AU 2001-296610	20011004	<--
AT 415158	T	20081215	AT 2001-977495	20011004	<--
US 20030229064	A1	20031211	US 2003-378206	20030303	<--
US 20050096304	A1	20050505	US 2004-922728	20040820	
US 20070232692	A1	20071004	US 2007-671823	20070206	

PRIORITY APPLN. INFO.:	US 1998-99390P	P	19980908
	US 1999-392122	A2	19990908
	US 2000-679932	A2	20001005
	US 2000-735205	A	20001212
	WO 2001-US31142	W	20011004
	US 2003-378206	A2	20030303

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

OTHER SOURCE(S): MARPAT 135:190400

AB Dithiocarbamate, particularly tetraethylthiuram disulfide, and thiocarbamate anions strongly inhibit the growth of cancer cells of a variety of cell types. Such inhibitory effect is enhanced by heavy metal ions such as copper ions, cytokines and ceruloplasmin. A method is presented for using tetraethylthiuram disulfide to reduce tumor growth, and to potentiate the effect of other anticancer agents. Chelates of disulfiram with a number of metal ions, including Cu2+, Zn2+, Ag1+, or Au3+ were synthesized. During generation of disulfiram-metal complexes, chelation of metal ions from the aqueous phase was suggested by a color change in the disulfiram-containing chloroform phase (from pale yellow to brilliant golden orange with complexation of gold ions). All metal complexes showed increased antiproliferative activity compared to disulfiram, but the most active compound was formed by the complex of gold with disulfiram, which was antiproliferative at nM concns.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L23 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER:	2001:338762 CAPLUS
DOCUMENT NUMBER:	134:362292
TITLE:	Methods of determining individual hypersensitivity to a pharmaceutical agent from gene expression profile
INVENTOR(S):	Farr, Spencer
PATENT ASSIGNEE(S):	Phase-1 Molecular Toxicology, USA
SOURCE:	PCT Int. Appl., 222 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001032928	A2	20010510	WO 2000-US30474	20001103 <--
WO 2001032928	A3	20020725		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-165398P P 19991105
US 2000-196571P P 20000411

AB The invention discloses methods, gene databases, gene arrays, protein arrays, and devices that may be used to determine the hypersensitivity of individuals to a given agent, such as drug or other chemical, in order to prevent toxic side effects. In one embodiment, methods of identifying hypersensitivity in a subject by obtaining a gene expression profile of multiple genes associated with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes associated with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes associated with hypersensitivity. The expression of the genes predetd. to be associated with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and apparatus useful for identifying hypersensitivity in a subject are also disclosed.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2001:185566 CAPLUS
DOCUMENT NUMBER: 134:217186
TITLE: Method of treating cancer using a thiuram disulfide such as tetraethyl thiuram disulfide
INVENTOR(S): Kennedy, Thomas Preston
PATENT ASSIGNEE(S): Charlotte-Mecklenburg Hospital Authority, USA
SOURCE: PCT Int. Appl., 60 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001017522	A1	20010315	WO 1999-US27193	19991115 <--

W: AU, CA, JP

RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,

PT, SE

US 20030065026	A1	20030403	US 1999-392122	19990908	<--
US 6589987	B2	20030708			
CA 2384059	A1	20010315	CA 1999-2384059	19991115	<--
EP 1214063	A1	20020619	EP 1999-963914	19991115	<--
EP 1214063	B1	20050727			

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY

JP 2003514769	T	20030422	JP 2001-521313	19991115	<--
AU 782029	B2	20050630	AU 2000-20255	19991115	<--
AT 300291	T	20050815	AT 1999-963914	19991115	<--
ES 2244237	T3	20051201	ES 1999-963914	19991115	<--

PRIORITY APPLN. INFO.:	US 1999-392122	A	19990908
	US 1998-99390P	P	19980908
	WO 1999-US27193	W	19991115

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB A dithiocarbamate, particularly tetra-Et thiuram disulfide, strongly inhibits the growth of cancer cells of a variety of cell types. Such inhibitory effect is enhanced by heavy metal ions (e.g. copper ions), cytokines, and ceruloplasmin. A method is presented for using tetra-Et thiuram disulfide to reduce tumor growth, and to potentiate the effect of other anticancer agents.

REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER:	2000:351162 CAPLUS
DOCUMENT NUMBER:	133:790
TITLE:	New use of glutamate antagonists for the treatment of cancer
INVENTOR(S):	Ikonomidou, Hrissanthi
PATENT ASSIGNEE(S):	Germany
SOURCE:	Eur. Pat. Appl., 21 pp. CODEN: EPXXDW
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
EP 1002535	A1	20000524	EP 1998-250380	19981028	<--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

AU 9964750	A	20000515	AU 1999-64750	19991022	<--
EP 1124553	A1	20010822	EP 1999-952622	19991022	<--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

JP 2002528415	T	20020903	JP 2000-578005	19991022	<--
EP 1586321	A1	20051019	EP 2005-12871	19991022	<--
EP 1586321	B1	20081210			

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY

EP 1649857	A2	20060426	EP 2005-12872	19991022	<--
EP 1649857	A3	20070328			

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY

AT 416769	T	20081215	AT 2005-12871	19991022	<--
US 6797692	B1	20040928	US 2001-830354	20010425	<--
US 20050054619	A1	20050310	US 2004-912159	20040806	
US 7247610	B2	20070724			
US 20050054650	A1	20050310	US 2004-912175	20040806	

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PRIORITY APPLN. INFO.:                      EP 1998-250380      A  19981028
                                            EP 1999-952622      A3 19991022
                                            WO 1999-EP8004      W  19991022
                                            US 2001-830354      A3 20010425
AB   New therapies can be devised based upon a demonstration of the role of
     glutamate in the pathogenesis of cancer.  Inhibitors of the
     interaction of glutamate with the AMPA, kainate, or NMDA receptor
     complexes are likely to be useful in treating cancer and can be
     formulated as pharmaceutical compns.  They can be identified by
     appropriate screens.
OS.CITING REF COUNT:     11     THERE ARE 11 CAPLUS RECORDS THAT CITE THIS
                                RECORD (11 CITINGS)
REFERENCE COUNT:          8     THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


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                 S DISULFIRAM/CN

     FILE 'REGISTRY' ENTERED AT 11:20:06 ON 10 MAY 2010
L2             1 S DISULFIRAM/CN

     FILE 'CAPLUS' ENTERED AT 11:20:06 ON 10 MAY 2010
L3          3380 S L2
                 S DISULFRAM/CN

     FILE 'REGISTRY' ENTERED AT 11:20:23 ON 10 MAY 2010
L4             0 S DISULFRAM/CN

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L5             0 S L4
L6          6756 S CURCUMIN

     FILE 'REGISTRY' ENTERED AT 11:20:42 ON 10 MAY 2010
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L8             1 S CURCUMIN/CN
L9             1 S BSO/CN
L10            1 S BCNU/CN

     FILE 'CAPLUS' ENTERED AT 11:21:30 ON 10 MAY 2010
L11         3380 S L7
L12         5428 S L8
L13         1947 S L9
L14         3851 S L10
L15      1004074 S CANCER OR TUMOR OR NEOPLASM
L16          224 S L11 AND L15
L17         1654 S L12 AND L15
L18            3 S L13 AND L15
L19         2789 S L14 AND L15
L20           34 S (L16 OR L17) AND (L18 OR L19)
L21           34 DUP REM L20 (0 DUPLICATES REMOVED)
L22           34 S L21
L23           10 S L21 AND AD<20030718

=> file medline embase biosis
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
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FULL ESTIMATED COST                                    42.74      91.95

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)       SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                 -8.50      -8.50

FILE 'MEDLINE' ENTERED AT 11:24:17 ON 10 MAY 2010

FILE 'EMBASE' ENTERED AT 11:24:17 ON 10 MAY 2010
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FILE 'BIOSIS' ENTERED AT 11:24:17 ON 10 MAY 2010
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=> d his

     (FILE 'HOME' ENTERED AT 11:19:38 ON 10 MAY 2010)

     FILE 'CAPLUS' ENTERED AT 11:19:51 ON 10 MAY 2010
L1          2300 S DISULFIRAM
                 S DISULFIRAM/CN

     FILE 'REGISTRY' ENTERED AT 11:20:06 ON 10 MAY 2010
L2             1 S DISULFIRAM/CN

     FILE 'CAPLUS' ENTERED AT 11:20:06 ON 10 MAY 2010
L3          3380 S L2
                 S DISULFRAM/CN

     FILE 'REGISTRY' ENTERED AT 11:20:23 ON 10 MAY 2010
L4             0 S DISULFRAM/CN

     FILE 'CAPLUS' ENTERED AT 11:20:23 ON 10 MAY 2010
L5             0 S L4
L6          6756 S CURCUMIN

     FILE 'REGISTRY' ENTERED AT 11:20:42 ON 10 MAY 2010
L7             1 S DISULFIRAM/CN
L8             1 S CURCUMIN/CN
L9             1 S BSO/CN
L10            1 S BCNU/CN

     FILE 'CAPLUS' ENTERED AT 11:21:30 ON 10 MAY 2010
L11         3380 S L7
L12         5428 S L8
L13         1947 S L9
L14         3851 S L10
L15      1004074 S CANCER OR TUMOR OR NEOPLASM
L16          224 S L11 AND L15
L17         1654 S L12 AND L15
L18            3 S L13 AND L15
L19         2789 S L14 AND L15
L20           34 S (L16 OR L17) AND (L18 OR L19)
L21           34 DUP REM L20 (0 DUPLICATES REMOVED)
L22           34 S L21
L23           10 S L21 AND AD<20030718

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:24:17 ON 10 MAY 2010

=> s l7<chem>

   SmartSELECT INITIATED
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New TRANSFER and ANALYZE Commands Now Available
See HELP TRANSFER and HELP ANALYZE for Details

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	3.33	95.28

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-8.50

FILE 'REGISTRY' ENTERED AT 11:24:40 ON 10 MAY 2010
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SET SMARTSELECT ON
SET COMMAND COMPLETED

SEL L7 1- CHEM
L24 SEL L7 1- CHEM : 72 TERMS

SET SMARTSELECT OFF
SET COMMAND COMPLETED

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	15.49	110.77

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-8.50

FILE 'MEDLINE' ENTERED AT 11:24:41 ON 10 MAY 2010

FILE 'EMBASE' ENTERED AT 11:24:41 ON 10 MAY 2010

FILE 'BIOSIS' ENTERED AT 11:24:41 ON 10 MAY 2010

S L24
2 FILES SEARCHED...
L25 53652 L24

=> s l8<chem>

SmartSELECT INITIATED
New TRANSFER and ANALYZE Commands Now Available
See HELP TRANSFER and HELP ANALYZE for Details

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	3.33	114.10

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-8.50

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FILE 'REGISTRY' ENTERED AT 11:25:20 ON 10 MAY 2010
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SET SMARTSELECT ON
SET COMMAND COMPLETED

SEL L8 1- CHEM
L26         SEL L8 1- CHEM :      42 TERMS

SET SMARTSELECT OFF
SET COMMAND COMPLETED


COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    15.49     129.59

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00      -8.50

FILE 'MEDLINE' ENTERED AT 11:25:20 ON 10 MAY 2010

FILE 'EMBASE' ENTERED AT 11:25:20 ON 10 MAY 2010
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FILE 'BIOSIS' ENTERED AT 11:25:20 ON 10 MAY 2010
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S L26
L27      18699 L26

=> s l9<chem>

   SmartSELECT INITIATED
New TRANSFER and ANALYZE Commands Now Available
See HELP TRANSFER and HELP ANALYZE for Details


COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     3.33     132.92

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00      -8.50

FILE 'REGISTRY' ENTERED AT 11:25:33 ON 10 MAY 2010
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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SET SMARTSELECT ON
SET COMMAND COMPLETED

SEL L9 1- CHEM
L28         SEL L9 1- CHEM :      13 TERMS

SET SMARTSELECT OFF
SET COMMAND COMPLETED
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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    15.49     148.41

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00      -8.50

FILE 'MEDLINE' ENTERED AT 11:25:33 ON 10 MAY 2010

FILE 'EMBASE' ENTERED AT 11:25:33 ON 10 MAY 2010
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FILE 'BIOSIS' ENTERED AT 11:25:33 ON 10 MAY 2010
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S L28
L29       5753 L28

=> s l10<chem>

   SmartSELECT INITIATED
New TRANSFER and ANALYZE Commands Now Available
See HELP TRANSFER and HELP ANALYZE for Details


COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                     3.33     151.74

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                     0.00      -8.50

FILE 'REGISTRY' ENTERED AT 11:25:41 ON 10 MAY 2010
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SET SMARTSELECT ON
SET COMMAND COMPLETED

SEL L10 1- CHEM
L30         SEL L10 1- CHEM :      21 TERMS

SET SMARTSELECT OFF
SET COMMAND COMPLETED


COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    15.49     167.23

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                     0.00      -8.50

FILE 'MEDLINE' ENTERED AT 11:25:41 ON 10 MAY 2010

FILE 'EMBASE' ENTERED AT 11:25:41 ON 10 MAY 2010
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FILE 'BIOSIS' ENTERED AT 11:25:41 ON 10 MAY 2010

S L30
L31 25542 L30

=> d his

(FILE 'HOME' ENTERED AT 11:19:38 ON 10 MAY 2010)

FILE 'CAPLUS' ENTERED AT 11:19:51 ON 10 MAY 2010
L1 2300 S DISULFIRAM
S DISULFIRAM/CN

FILE 'REGISTRY' ENTERED AT 11:20:06 ON 10 MAY 2010
L2 1 S DISULFIRAM/CN

FILE 'CAPLUS' ENTERED AT 11:20:06 ON 10 MAY 2010
L3 3380 S L2
S DISULFRAM/CN

FILE 'REGISTRY' ENTERED AT 11:20:23 ON 10 MAY 2010
L4 0 S DISULFRAM/CN

FILE 'CAPLUS' ENTERED AT 11:20:23 ON 10 MAY 2010
L5 0 S L4
L6 6756 S CURCUMIN

FILE 'REGISTRY' ENTERED AT 11:20:42 ON 10 MAY 2010
L7 1 S DISULFIRAM/CN
L8 1 S CURCUMIN/CN
L9 1 S BSO/CN
L10 1 S BCNU/CN

FILE 'CAPLUS' ENTERED AT 11:21:30 ON 10 MAY 2010
L11 3380 S L7
L12 5428 S L8
L13 1947 S L9
L14 3851 S L10
L15 1004074 S CANCER OR TUMOR OR NEOPLASM
L16 224 S L11 AND L15
L17 1654 S L12 AND L15
L18 3 S L13 AND L15
L19 2789 S L14 AND L15
L20 34 S (L16 OR L17) AND (L18 OR L19)
L21 34 DUP REM L20 (0 DUPLICATES REMOVED)
L22 34 S L21
L23 10 S L21 AND AD<20030718

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:24:17 ON 10 MAY 2010

FILE 'REGISTRY' ENTERED AT 11:24:40 ON 10 MAY 2010
SET SMARTSELECT ON
L24 SEL L7 1- CHEM : 72 TERMS
SET SMARTSELECT OFF

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:24:41 ON 10 MAY 2010
L25 53652 S L24

FILE 'REGISTRY' ENTERED AT 11:25:20 ON 10 MAY 2010

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                SET SMARTSELECT ON
L26         SEL L8 1- CHEM :      42 TERMS
                SET SMARTSELECT OFF

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:25:20 ON 10 MAY 2010
L27       18699 S L26

     FILE 'REGISTRY' ENTERED AT 11:25:33 ON 10 MAY 2010
                SET SMARTSELECT ON
L28         SEL L9 1- CHEM :      13 TERMS
                SET SMARTSELECT OFF

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:25:33 ON 10 MAY 2010
L29        5753 S L28

     FILE 'REGISTRY' ENTERED AT 11:25:41 ON 10 MAY 2010
                SET SMARTSELECT ON
L30         SEL L10 1- CHEM :      21 TERMS
                SET SMARTSELECT OFF

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:25:41 ON 10 MAY 2010
L31       25542 S L30

=> s cancer or tumor or neoplasm
L32    6349459 CANCER OR TUMOR OR NEOPLASM

=> s l25 and l32
L33       5417 L25 AND L32

=> s l27 and l32
L34       6435 L27 AND L32

=> s l29 and l32
L35       2099 L29 AND L32

=> s l31 and l32
L36      18174 L31 AND L32

=> s (l33 or l34) and (l35 or l36)
L37         66 (L33 OR L34) AND (L35 OR L36)

=> s l37 and pd<20030718
   1 FILES SEARCHED...
L38         22 L37 AND PD<20030718

=> d l38 1-22 ibib abs

L38  ANSWER 1 OF 22     MEDLINE on STN
ACCESSION NUMBER:   2002705766     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 12467214
TITLE:              Disulfiram induces apoptosis in human melanoma
                    cells: a redox-related process.
AUTHOR:             Cen Dazhi; Gonzalez Rachel I; Buckmeier Julie A; Kahlon
                    Ravi S; Tohidian Nilou B; Meyskens Frank L Jr
CORPORATE SOURCE:   Department of Medicine, Chao Family Comprehensive Cancer
                    Center, College of Medicine, University of California,
                    Irvine, 101 City Drive South, Building 23, Suite 403,
                    Orange, CA 92868, USA.
CONTRACT NUMBER:    P30CA62203 (United States NCI NIH HHS)
SOURCE:             Molecular cancer therapeutics, (2002 Jan) Vol. 1,
                    No. 3, pp. 197-204.
                    Journal code: 101132535. ISSN: 1535-7163. L-ISSN:
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1535-7163.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200301
ENTRY DATE: Entered STN: 17 Dec 2002
Last Updated on STN: 17 Jan 2003
Entered Medline: 16 Jan 2003

AB Melanoma is highly resistant to conventional chemotherapy. We have demonstrated that redox regulation in melanoma cells is aberrant, and redox-modulating agents can induce cell apoptosis. We have currently explored the effect of disulfiram (DSF), a member of the dithiocarbamate family, on apoptosis of melanoma cells in vitro. Human metastatic melanoma cells c81-46A, c81-61, and c83-2C were treated with DSF and apoptosis measured. DSF, at a dose of 25-50 ng/ml, consistently caused a 4-6-fold increase in apoptosis. The same dose of DSF did not significantly affect apoptosis in melanocytes. Coincubation of N-acetyl-cysteine reversed the DSF-induced apoptosis. Buthionine sulfoximine (BSO), an inhibitor of gamma-glutamyl-cysteine synthetase, as a single agent caused a approximately 2-fold increase in apoptosis when incubated with melanoma cells for 4 days. BSO slightly enhanced the level of apoptosis induced by DSF (4-10% higher than DSF alone). Intracellular glutathione was remarkably depleted with BSO treatment. DSF did not cause glutathione depletion; however, the ratio of reduced and oxidized glutathione was significantly decreased (14% of control), and N-acetyl-cysteine partially restored the ratio to 30% of control. There was a transient (2-fold) elevation of intracellular superoxide level after 24 h of DSF treatment (before the overt apoptosis). The intracellular H2O2 level progressively decreased with time. DSF decreased the mitochondrial membrane polarization in a time-dependent manner, and there was a significant inverse correlation between apoptosis and mitochondrial membrane polarization. We propose that DSF-induced apoptosis is redox related but involves a different mechanism from BSO-induced apoptosis in tumor cells. Our findings have provided new data for additional understanding of drug-induced apoptosis in melanoma cells and suggests an alternative therapeutic approach to melanoma.

L38 ANSWER 2 OF 22 MEDLINE on STN
ACCESSION NUMBER: 2002187475 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11920175
TITLE: The impact of autologous stem cell transplantation on the prognosis of mantle cell lymphoma: a joint analysis of two prospective studies with 46 patients.
AUTHOR: Dreger P; Martin S; Kuse R; Sonnen R; Glass B; Kroger N; Parwaresch R; Kneba M; Schmitz N; Haas R
CORPORATE SOURCE: Second Department of Medicine, University of Kiel, Germany.
SOURCE: The hematology journal : the official journal of the European Haematology Association / EHA, (2000) Vol. 1, No. 2, pp. 87-94.
Journal code: 100965523. ISSN: 1466-4860. L-ISSN: 1466-4860.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
(COMPARATIVE STUDY)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)

(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 3 Apr 2002
Last Updated on STN: 9 May 2002
Entered Medline: 8 May 2002

AB INTRODUCTION: The purpose of this analysis was to investigate if early sequential high-dose therapy with autologous stem cell transplantation (ASCT) can improve the poor prognosis of patients with disseminated mantle cell lymphoma (MCL). PATIENTS AND METHODS: A joint analysis of two parallel single center studies was performed. Both were characterized by a sequential high-dose therapy consisting of an intensive chemotherapy ('HAM' or 'Dexa-BEAM') for mobilization of peripheral blood stem cells and induction of minimal disease followed by a total body irradiation-containing myeloablative regimen and ASCT. Forty-six patients with reference panel-confirmed stage III/IV MCL were included. Thirty-four patients were accrued to the protocol immediately after diagnosis ('upfront ASCT' group). These 34 patients received a standard first-line regimen prior to mobilization. The remaining 12 patients were put on the protocol later during the course of their disease ('delayed ASCT' group). RESULTS: All patients were in remission after mobilization chemotherapy and proceeded to ASCT; there were no exclusions due to poor response, poor mobilization, or patient refusal. With a follow-up of 24 (2-73) months post transplant, the event-free and overall survival probabilities at 2 years were 77 and 100% for the upfront ASCT group compared to 30% (P=0.0007) and 54% (P=0.0016) for the delayed ASCT group. Event-free and overall survival tended to be longer in the upfront ASCT group than in the delayed ASCT group also if calculated from initial diagnosis (76 and 93% vs 42 and 63%, respectively, at 4 years after diagnosis; median follow-up 35 months), although this was not statistically significant. Besides timing of ASCT, only spleen size was identified as an independent predictor of survival by univariate and multivariate analysis. CONCLUSION: ASCT is not curative but may improve the prognosis of patients with MCL if performed as part of an intensive first-line treatment strategy. In contrast, the benefits of this approach for salvaging individuals with relapsed disease appear to be limited.

L38 ANSWER 3 OF 22 MEDLINE on STN
ACCESSION NUMBER: 2001548694 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11595684
TITLE: Sequential tumor biopsies in early phase clinical trials of anticancer agents for pharmacodynamic evaluation.
AUTHOR: Dowlati A; Haaga J; Remick S C; Spiro T P; Gerson S L; Liu L; Berger S J; Berger N A; Willson J K
CORPORATE SOURCE: Division of Hematology/Oncology, Department of Medicine, Ireland Cancer Center at University Hospitals of Cleveland, 11100 Euclid Avenue, Cleveland, OH 44106, USA.. axd44@po.cwru.edu
CONTRACT NUMBER: M01-RR-00080 (United States NCRR NIH HHS)
P30 CA43703 (United States NCI NIH HHS)
U01 CA62502 (United States NCI NIH HHS)
SOURCE: Clinical cancer research : an official journal of the American Association for Cancer Research, (2001 Oct) Vol. 7, No. 10, pp. 2971-6.
Journal code: 9502500. ISSN: 1078-0432. L-ISSN: 1078-0432.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112

ENTRY DATE: Entered STN: 15 Oct 2001
Last Updated on STN: 22 Jan 2002
Entered Medline: 5 Dec 2001

AB PURPOSE: In the setting of target-based anticancer drug development, it is critical to establish that the observed preclinical activity can be attributed to modulation of the intended target in early phase trials in human subjects. This paradigm of target modulation allows us to determine a Phase II or III dose (optimal biochemical/biological modulatory dose) that may not necessarily be the maximum tolerated dose. A major obstacle to target-based (often cytostatic) drug development has been obtaining relevant tumor tissue during clinical trials of these novel agents for laboratory analysis of the putative marker of drug effect. EXPERIMENTAL DESIGN: From 1989 to present, we have completed seven clinical trials in which the end point was a biochemical or biological modulatory dose in human tumor tissues (not surrogate tissue). Eligibility enrollment required that patients have a biopsiable lesion either with computerized tomography (CT) guidance or direct visualization and consent to sequential (pre and posttreatment) biopsies. RESULTS: A total of 192 biopsies were performed in 107 patients. All but 8 patients had sequential pre and posttreatment biopsies. Seventy-eight (73%) of the 107 patients had liver lesion biopsies. In eight patients, either one or both biopsies contained insufficient viable tumor tissue or no tumor tissue at all for analysis. Of a total of 99 patients in whom we attempted to obtain paired biopsies, a total of 87 (88%) were successful. Reasons for failure included patient refusal for a second biopsy (n = 2), vasovagal reaction with first biopsy precluding a second biopsy (n = 1), subcapsular hepatic bleeding (n = 1), and most commonly obtaining necrotic tumor, fibrous, or normal tissue in one of the two sequential biopsies (n = 8). CONCLUSIONS: This is the first and largest reported series demonstrating that with adequate precautions and experience, sequential tumor biopsies are feasible and safe during early phase clinical trials.

L38 ANSWER 4 OF 22 MEDLINE on STN
ACCESSION NUMBER: 1997178737 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9053470
TITLE: Intensified and high-dose chemotherapy with granulocyte colony-stimulating factor and autologous stem-cell transplantation support as first-line therapy in high-risk diffuse large-cell lymphoma.
AUTHOR: Vitolo U; Cortellazzo S; Liberati A M; Freilone R; Falda M; Bertini M; Botto B; Cinieri S; Levis A; Locatelli F; Lovisone E; Marmont F; Pizzuti M; Rossi A; Viero P; Barbui T; Grignani F; Resegotti L
CORPORATE SOURCE: Divisione di Ematologia Azienda Ospedaliera S. Giovanni Battista, Torino, Italy.
SOURCE: Journal of clinical oncology : official journal of the American Society of Clinical Oncology, (1997 Feb) Vol. 15, No. 2, pp. 491-8.
Journal code: 8309333. ISSN: 0732-183X. L-ISSN: 0732-183X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 21 Mar 1997
Last Updated on STN: 21 Mar 1997
Entered Medline: 10 Mar 1997

AB PURPOSE: In our previous study with MACOPB, we identified a high-risk group of patients with a poor 3-year survival rate of 29%. These patients were defined as having at diagnosis advanced-stage disease with high tumor burden (TB) and elevated lactate dehydrogenase (LDH) level

or bone marrow (BM) involvement. A novel therapeutic scheme was investigated to improve the outcome of these patients. PATIENTS AND METHODS: Fifty patients with high-risk diffuse large-cell lymphoma (DLCL) were enrolled. The therapeutic scheme includes three phases: induction with 8 weeks of MACOPB; intensification with a 3-day course of mitoxantrone 8 mg/m2 plus high-dose cytarabine (HDARA-C) 2 g/m2 every 12 hours plus dexamethasone 4 mg/m2 every 12 hours (MAD protocol) and granulocyte colony-stimulating factor (G-CSF) 5 microg/kg on days 4 to 17 to harvest peripheral-blood progenitor cells (PBPC); consolidation with carmustine (BCNU), etoposide, ARA-C, and melphalan (BEAM) regimen; plus autologous stem-cell transplantation (ASCT) with PBPC, marrow, or both. RESULTS: Thirty-six patients (72%) achieved a complete response (CR), 11 (22%) showed no response (NR), and three (6%) died of toxicity. Among the 22 PRs or NRs after the induction phase, 56% of patients achieved a CR with subsequent intensified therapy. With a median follow-up duration of 32 months, the overall survival and failure-free survival rates were 56% and 50%, respectively. The disease-free survival rate is 69% at 32 months. Leukapheresis after MAD and G-CSF yielded a median of 32 x 10(6)/kg CD34+ cells and 80 x 10(4)/kg granulocyte-macrophage colony-forming units (CFU-GM). Thirty-nine patients were autografted and 11 did not undergo ASCT: six because of disease progression, four due to toxicity, and one because of patient refusal. The median times to achieve engrafment were 11 days (range, 7 to 19) to a neutrophil count greater than 0.5 x 10(9)/L and 12 days (range, 8 to 60) to a platelet count greater than 50 x 10(9)/L. CONCLUSION: This sequential scheme with intensified and high-dose chemotherapy with ASCT is feasible with moderate toxicity and may improve the outcome in high-risk DLCL.

L38 ANSWER 5 OF 22 MEDLINE on STN
ACCESSION NUMBER: 1985289898 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3928682
TITLE: Hydrogen peroxide from cellular metabolism of cystine. A requirement for lysis of murine tumor cells by vernolepin, a glutathione-depleting antineoplastic.
AUTHOR: Arrick B A; Griffo W; Cohn Z; Nathan C
CONTRACT NUMBER: CA22090 (United States NCI NIH HHS)
HL-07029 (United States NHLBI NIH HHS)
SOURCE: The Journal of clinical investigation, (1985 Aug) Vol. 76, No. 2, pp. 567-74.
Journal code: 7802877. ISSN: 0021-9738. L-ISSN: 0021-9738.
Report No.: NLM-PMC423862.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198510
ENTRY DATE: Entered STN: 20 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 2 Oct 1985

AB The sesquiterpene lactone antineoplastic vernolepin acutely depletes murine tumor cell glutathione (GSH), and lyses the cells by an unknown mechanism that is enhanced synergistically by inhibition of GSH synthesis with buthionine sulfoximine (BSO) (Arrick et al. 1983. J. Clin. Invest. 71:258). We found here that lysis of P815 mastocytoma cells by vernolepin, with or without BSO, required cystine in the culture medium. Addition of catalase markedly suppressed vernolepin-mediated cytolysis in cystine-containing media, suggesting the involvement of hydrogen peroxide in the cytolytic action of vernolepin. Consistent with this, inhibition of tumor cell glutathione

disulfide reductase with 1,3-bis(2
-chloroethyl)-1-nitrosourea or inhibition of
endogenous catalase with aminotriazole synergistically augmented cytolysis
by vernolepin. Moreover, H2O2 was released by suspensions of P815 cells
in cystine-containing buffer (63 pmol/10(6) cells X h). Omission of
cystine reduced the rate of H2O2 accumulation 10-fold. No H2O2 was
detected without cells. Cytolysis by vernolepin could be restored in
cystine-deficient medium by several other disulfides, themselves
noncytolytic, such as disulfiram and oxidized Captopril, as well
as by cysteine. In contrast, withholding two other essential amino acids
(leucine or tryptophan) or adding cycloheximide did not interfere with
cytolysis by vernolepin. These results suggest that cellular uptake of
disulfides of physiologic and pharmacologic interest may be followed by
their intracellular reduction and autooxidation with generation of H2O2.
This previously unrecognized source of intracellular oxidant stress may be
an important component of injury to GSH-depleted cells.

L38 ANSWER 6 OF 22 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights
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ACCESSION NUMBER: 2002405422 EMBASE
TITLE: Brain cancer: A case of glioblastoma multiforme.
AUTHOR: Chang, Raymond, Dr. (correspondence); Finlay, Jonathan;
Badmaev, Vladimir; Singh, Ram Harsh; Chapman, Jnani
CORPORATE SOURCE: Institute of East-West Medicine, 102 East 30th Street, New
York, NY 10016, United States. rchang@eastwestmed.org
SOURCE: Journal of Alternative and Complementary Medicine, (
Oct 2002) Vol. 8, No. 5, pp. 551-558.
Refs: 5
ISSN: 1075-5535 CODEN: JACPFP
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Dec 2002
Last Updated on STN: 2 Dec 2002

L38 ANSWER 7 OF 22 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights
reserved on STN
ACCESSION NUMBER: 2002119492 EMBASE
TITLE: Redox signaling-mediated regulation of
lipopolysaccharide-induced proinflammatory cytokine
biosynthesis in alveolar epithelial cells.
AUTHOR: Haddad, John J., Dr. (correspondence); Land, Stephen C.
CORPORATE SOURCE: Neuroscience Research Laboratory, Department of Anesthesia,
Univ. of California at San Francisco, 513 Parnassus Avenue,
San Francisco, CA 94143-0542, United States. haddadj@anesth
esia.ucsf.edu
SOURCE: Antioxidants and Redox Signaling, (2002) Vol. 4,
No. 1, pp. 179-193.
Refs: 46
ISSN: 1523-0864 CODEN: ARSIF2
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical and Experimental Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 18 Apr 2002
Last Updated on STN: 18 Apr 2002
AB The regulation of cytokine gene transcription and biosynthesis involves

the reduction-oxidation (redox)-sensitive nuclear factor-κB (NF-κB), whose activation is mediated by an upstream kinase that regulates the phosphorylation of inhibitory-κB (IκB). It was hypothesized that lipopolysaccharide (LPS)-induced biosynthesis of interleukin-1β, interleukin-6, and tumor necrosis factor-α in vitro is regulated by redox equilibrium. In alveolar epithelial cells, we investigated the role of L-buthionine-(S,R)-sulfoximine (BSO), an irreversible inhibitor of γ-glutamylcysteine synthetase, the rate-limiting enzyme in GSH biosynthesis, 1,3-bis-(2-chloroethyl)-1-nitrosourea (BCNU), which inhibits glutathione oxidized disulfide reductase, pyrrolidine dithiocarbamate (PDTC), an antioxidant/prooxidant thiuram, and N-acetyl-L-cysteine (NAC), an antioxidant and GSH precursor, in regulating LPS-induced cytokine biosynthesis and IκB-α/NF-κB signaling. BSO blockaded the phosphorylation of IκB-α, reduced its degradation, and inhibited NF-κB activation, besides augmenting LPS-mediated biosynthesis of cytokines. BCNU up-regulated LPS-induced release of cytokines, an effect associated with partial phosphorylation/degradation of IκB-α and inhibition of the DNA binding activity. PDTC, which partially affected LPS-induced IκB-α phosphorylation/degradation, otherwise blockading NF-κB activation, reduced LPS-dependent up-regulation of cytokine release. Pretreatment with BSO did not abolish the NAC-dependent reduction of LPS-induced cytokine release, despite the fact that NAC marginally amplified IκB-α phosphorylation/degradation and suppressed NF-κB activation. These results indicate that cytokines are redox-sensitive mediators and that the IκB-α/NF-κB pathway is redox-sensitive and differentially implicated in mediating redox-dependent regulation of LPS-induced release of proinflammatory cytokines.

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ACCESSION NUMBER: 2001403840 EMBASE
TITLE: Autologous transplantation in acute myeloid leukemia: Peripheral blood stem cell harvest after mobilization in steady state by granulocyte colony-stimulating factor alone.
AUTHOR: Voog, E.; Le, Q.H.; Philip, I.; Benetaib, B.; Michallet, M.; Fiere, D.; Thomas, X. (correspondence)
CORPORATE SOURCE: Service d'Hematologie, Service des Maladies du Sang, Hopital E. Herriot, 69437 Lyon, Cedex 03, France. xavier.thomas@chu-lyon.fr
SOURCE: Annals of Hematology, (2001) Vol. 80, No. 10, pp. 584-591.
Refs: 43
ISSN: 0939-5555 CODEN: ANHEE8
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
025 Hematology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 6 Dec 2001
Last Updated on STN: 6 Dec 2001

AB In order to determine whether granulocyte colony-stimulating factor (G-CSF) alone initiated during steady state was able to mobilize peripheral blood stem cells (PBSC) in acute myeloid leukemia (AML) and to assess predictive factors for engraftment after autologous PBSC

transplantation, we studied 49 successive adult AML patients for whom autologous transplantation was planned between July 1994 and November 1998. G-CSF was used as priming agent and was initiated at least 4 weeks after the last day of chemotherapy, while neutrophil count was >0.5+10 9/1 and platelet count was >30+109/1. A median of three aphereses was performed resulting in a median collection of 14.8+108 nucleated cells/kg containing 7.7+108 mononuclear cells/kg, 47.1+104 CFU-GM/kg, and 3.8+106 CD34+ cells/kg. A significant correlation was observed between nucleated cell, mononuclear cell, and CFU-GM yields, while no correlation was found with CD34+ cell yield. Recruitment was not significantly different in patients with CD34+ leukemic cells at the time of initial diagnosis when compared to that of those presenting with CD34- blastic cells. Thirty-three patients actually underwent transplantation. Reasons for not autografting were inadequate stem cell harvest (ten patients), early relapse (two patients), prolonged neutropenia (one patient), organ failure (two patients), or patient refusal (one patient). Median time to achieve a neutrophil count greater than 0.5+109/1 and platelet count >50+109/1 untransfused was 13 and 36 days, respectively. A predictive factor for a shorter period neutropenia and a shorter thrombopenia was a higher count of harvested nucleated cells ($p<0.01$ and $p=0.02$, respectively). A higher count of harvested cells was also a predictive factor for less red cell and platelet transfusions ($p=0.03$ and $p=0.02$, respectively). The number of CD34+ harvested PBSC was not predictive for engraftment. We conclude that PBSC mobilization with G-CSF alone initiated in steady state is a feasible, safe, and suitable procedure for harvesting cells in sight of autologous transplantation in adult acute myeloid leukemia.

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ACCESSION NUMBER: 2001213017 EMBASE

TITLE: High-dose therapy and autologous stem-cell transplantation versus conventional-dose consolidation/maintenance therapy as postremission therapy for adult patients with lymphoblastic lymphoma: Results of a randomized trial of the european group for blood and marrow transplantation and the united kingdom lymphoma group.

AUTHOR: Sweetenham, J.W., Dr. (correspondence); Santini, G.; Qian, W.; Guelfi, M.; Schmitz, N.; Simnett, S.; Nagler, A.; Holte, H.; Kvaloy, S.; Bruzzi, P.; Goldstone, A.H.

CORPORATE SOURCE: Univ. of Colorado Hlth. Sci. Center, Division of Medical Oncology-B171, 4200 E 9th Ave., Denver, CO 80262, United States. john.sweetenham@uchsc.edu

SOURCE: Journal of Clinical Oncology, (1 Jun 2001) Vol. 19, No. 11, pp. 2927-2936.
Refs: 21
ISSN: 0732-183X CODEN: JCONDN

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer
025 Hematology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 17 Jul 2001
Last Updated on STN: 17 Jul 2001

AB Purpose: To determine whether a combination of high-dose therapy and autologous stem-cell transplantation (ASCT) is superior to conventional-dose consolidation and maintenance chemotherapy as postremission therapy in adults with lymphoblastic lymphoma. Patients and Methods: One hundred nineteen patients were entered onto this prospective randomized trial from 37 centers. Patients received standard remission

induction therapy, and responding patients were randomized either to continue with a conventional consolidation/maintenance protocol (CC) or to receive high-dose therapy and ASCT. In some centers, patients with HLA-identical sibling donors were registered on the trial but proceeded to allogeneic bone marrow transplantation (BMT) without randomization. Results: Of the 119 patients entered, 111 were assessable for response to induction therapy. The overall response rate was 82% (56% complete response, 26% partial response). Of the 98 patients eligible for randomization, 65 were randomized, 31 to ASCT and 34 to CC. Reasons for failure to randomize included patient refusal (12 patients), early progression or death on induction therapy (eight patients), excessive toxicity of induction regimen (six patients), and elective allogeneic BMT (12 patients). With a median follow-up of 37 months, the actuarial 3-year relapse-free survival rate is 24% for the CC arm and 55% for the ASCT arm (hazards ratio = 0.55 in favor of the ASCT arm; 95% confidence interval [CI], 0.29 to 1.04; P = .065). The corresponding figures for overall survival are 45% and 56%, respectively (hazards ratio = 0.87 in favor of the ASCT arm; 95% CI, 0.42 to 1.81; P = .71). Conclusion: The use of ASCT in adults with lymphoblastic lymphoma in first remission produced a trend for improved relapse-free survival but did not improve overall survival compared with conventional-dose therapy in this small randomized trial. .COPYRGT. 2001 by American Society of Clinical Oncology.

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ACCESSION NUMBER: 1997050546 EMBASE

TITLE: Intensified and high-dose chemotherapy with granulocyte colony- stimulating factor and autologous stem-cell transplantation support as first- line therapy in high-risk diffuse large-cell lymphoma.

AUTHOR: Vitolo, Umberto, Dr. (correspondence)

CORPORATE SOURCE: Divisione di Ematologia, Azienda Ospedaliera S. Giovanni B., corso Bramante 90, 10126 Torino, Italy.

AUTHOR: Cortellazzo, Sergio; Liberati, Anna Maria; Freilone, Roberto; Falda, Michele; Bertini, Marilena; Botto, Barbara; Cinieri, Saverio; Levis, Alessandro; Locatelli, Franco; Lovisone, Elisabetta; Marmont, Filippo; Pizzuti, Michele; Rossi, Andrea; Viero, Piera; Barbui, Tiziano; Grignani, Fausto; Resegotti, Luigi

AUTHOR: Vitolo, Umberto, Dr. (correspondence)

CORPORATE SOURCE: Divisione di Ematologia, AOSGBM, carso Brarnante 90, 10126 Torino, Italy.

SOURCE: Journal of Clinical Oncology, (Feb 1997) Vol. 15, No. 2, pp. 491-498.
Refs: 38
ISSN: 0732-183X CODEN: JCONDN

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer
025 Hematology
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 10 Mar 1997
Last Updated on STN: 10 Mar 1997

AB Purpose: In our previous study with MACOPB, we identified a high-risk group of patients with a poor 3-year survival rate of 29%. These patients were defined as having at diagnosis advanced-stage disease with high tumor burden (TB) and elevated lactate dehydrogenase (LDH) level or bone marrow (BM) involvement. A novel therapeutic scheme was

investigated to improve the outcome of these patients. Patients and Methods: Fifty patients with high- risk diffuse large-cell lymphoma (DLCL) were enrolled. The therapeutic scheme includes three phases: induction with 8 weeks of MACOPB; intensification with a 3-day course of mitoxantrone 8 mg/m2 plus high-dose cytarabine (HDARA-C) 2 g/m2 every 12 hours plus dexamethasone 4 mg/m2 every 12 hours (MAD protocol) and granulocyte colony-stimulating factor (G-CSF) 5 μg/kg on days 4 to 17 to harvest peripheral-blood progenitor cells (PBPC); consolidation with carmustine (BCNU), etoposide, ARA-C, and melphalan (BEAM) regimen; plus autologous stem-cell transplantation (ASCT) with PBPC, marrow, or both. Results: Thirty-six patients (72%) achieved a complete response (CR), 11 (22%) showed no response (NR), and three (6%) died of toxicity. Among the 22 PRs or NRs after the induction phase, 56% of patients achieved a CR with subsequent intensified therapy. With a median follow-up duration of 32 months, the overall survival and failure-free survival rates were 56% and 50%, respectively. The disease-free survival rate is 69% at 32 months. Leukapheresis after MAD and G-CSF yielded a median of 32 x 106/kg CD34+ cells and 80 x 104/kg granulocyte-macrophage colony-forming units (CFU-GM). Thirty-nine patients were autografted and 11 did not undergo ASCT: six because of disease progression, four due to toxicity, and one because of patient refusal. The median times to achieve engraftment were 11 days (range, 7 to 19) to a neutrophil count greater than 0.5 x 109/L and 12 days (range, 8 to 60) to a platelet count greater than 50 x 109/L. Conclusion: This sequential scheme with intensified and high-dose chemotherapy with ASCT is feasible with moderate toxicity and may improve the outcome in high-risk DLCL.

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ACCESSION NUMBER: 1996018010 EMBASE
TITLE: Outcome of extensive evaluation before adjuvant therapy in women with breast cancer and 10 or more positive axillary lymph nodes.
AUTHOR: Crump, Michael, Dr. (correspondence)
CORPORATE SOURCE: Toronto Hospital, 200 Elizabeth St, Toronto, Ont. M5G 2C4, Canada.
AUTHOR: Goss, Paul E.; Prince, Miles; Girouard, Caroline
AUTHOR: Crump, Michael, Dr. (correspondence)
CORPORATE SOURCE: Toronto Hospital, Mulock-Larkin Wing 2-018, 200 Elizabeth St, Toronto, Ont. M5G 2C4, Canada.
SOURCE: Journal of Clinical Oncology, (Jan 1996) Vol. 14, No. 1, pp. 66-69.
Refs: 21
ISSN: 0732-183X CODEN: JCONDN
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 6 Feb 1996
Last Updated on STN: 6 Feb 1996

AB Purpose: To evaluate the effect of extensive screening of women with high-risk, node-positive breast cancer on the detection of occult metastatic disease and patient eligibility for a randomized trial of the addition of high-dose chemotherapy and autologous bone marrow support (ABMT) to standard adjuvant therapy. Patients and Methods: Women with resected T1-3N1,2 primary breast cancer and ≥ 10 positive axillary lymph nodes referred for possible trial participation were evaluated for this report. All had normal chest x- ray, bone scan, and liver ultrasound performed by the referring physician. Those who provided

informed consent for the randomized trial were further evaluated according to protocol with computed tomographic (CT) scans of the head, chest, abdomen, and pelvis and bilateral bone marrow biopsies; those with metastatic disease detected by any of these tests were excluded from study registration. Results: Forty-four women were evaluated between February 1993 and April 1995. Fourteen did not undergo further protocol staging because of refusal to participate or the presence of metastatic disease on clinical assessment or review of outside radiologic studies. The remaining 30 underwent additional investigations, and seven (23%; 95% confidence interval [CI], 12% to 41%) bad metastases detected by CT scanning (four patients) or bone marrow biopsy (three patients) not detected by routine screening. Conclusion: Although the number of patients evaluated is small, these data suggest that some of the improvement in outcome of women with ≥ 10 positive axillary lymph nodes who receive ABMT as part of adjuvant chemotherapy in phase II trials may be from the exclusion of patients with occult metastatic disease. The importance of these exclusions can only be determined by ongoing, randomized controlled trials.

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ACCESSION NUMBER: 1985227070 EMBASE

TITLE: Hydrogen peroxide from cellular metabolism of cystine: A requirement for lysis of murine tumor cells by vernolepin, a glutathione-depleting antineoplastic.

AUTHOR: Arrick, B.A.; Griffo, W.; Cohn, Z.; Nathan, C.

CORPORATE SOURCE: The Rockefeller University, New York, NY 10021, United States.

SOURCE: Journal of Clinical Investigation, (1985) Vol. 76, No. 2, pp. 567-574.
ISSN: 0021-9738 CODEN: JCINAO

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer
029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index

LANGUAGE: English

ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB The sesquiterpene lactone antineoplastic vernolepin acutely depletes murine tumor cell glutathione (GSH), and lyses the cells by an unknown mechanism that is enhanced synergistically by inhibition of GSH synthesis with buthionine sulfoximine (BSO). We found here that lysis of P815 mastocytoma cells by vernolepin, with or without BSO , required cystine in the culture medium. Addition of catalase markedly suppressed vernolepin-mediated cytolysis in cystine-containing media, suggesting the involvement of hydrogen peroxide in the cytolytic action of vernolepin. Consistent with this, inhibition of tumor cell glutathione disulfide reductase with 1,3-bis (2-chloroethyl)-1-nitrosourea or inhibition of endogenous catalase with aminotriazole synergistically augmented cytolysis by vernolepin. Moreover, H2O2 was released by suspensions of P815 cells in cystine-containing buffer (63 pmol/106 cells .ovrhdot. h). Omission of cystine reduced the rate of H2O2 accumulation 10-fold. No H2O2 was detected without cells. Cytolysis by vernolepin could be restored in cystine-deficient medium by several other disulfides, themselves non-cytolytic, such as disulfiram and oxidized Captopril, as well as by cysteine. In contrast, withholding two other essential amino acids (leucine or tryptophan) or adding cycloheximide did not interfere with cytolysis by vernolepin. These results suggest that cellular uptake of disulfides of physiologic and pharmacologic interest

may be followed by their intracellular reduction and autooxidation with generation of H2O2. This previously unrecognized source of intracellular oxidant stress may be an important component of injury to GSH-depleted cells.

L38 ANSWER 13 OF 22 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1984057569 EMBASE
TITLE: Elmustine.
SOURCE: Drugs of the Future, (1984) Vol. 9, No. 1, pp. 18-19.
ISSN: 0377-8282 CODEN: DRFUD4
COUNTRY: Spain
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

L38 ANSWER 14 OF 22 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1979134140 EMBASE
TITLE: Carcinoma of the colon: Epidemiology, etiology, diagnosis, and treatment.
AUTHOR: Diggs, C.H.
CORPORATE SOURCE: Baltimore Cancer Res. Cent., Univ. Maryland Sch. Med., Baltimore, Md. 21201, United States.
SOURCE: American Journal of the Medical Sciences, (1979) Vol. 277, No. 1, pp. 4-16.
ISSN: 0002-9629 CODEN: AJMSA9
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 006 Internal Medicine
005 General Pathology and Pathological Anatomy
048 Gastroenterology
009 Surgery
037 Drug Literature Index
017 Public Health, Social Medicine and Epidemiology
016 Cancer
020 Gerontology and Geriatrics
LANGUAGE: English
AB A short survey of epidemiology, etiology, diagnosis and treatment of carcinoma of the colon is given. Literature review.

L38 ANSWER 15 OF 22 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 0012467214 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: Disulfiram induces apoptosis in human melanoma cells: a redox-related process..
AUTHOR: Cen, Dazhi (correspondence); Gonzalez, Rachel I; Buckmeier, Julie A; Kahlon, Ravi S; Tohidian, Nilou B; Meyskens Jr., Frank L
CORPORATE SOURCE: Department of Medicine, Chao Family Comprehensive Cancer Center, College of Medicine, University of California, Irvine, 101 City Drive South, Building 23, Suite 403, Orange, CA 92868, USA..
SOURCE: Molecular cancer therapeutics, (Jan 2002) Vol. 1, No. 3, pp. 197-204.
ISSN: 1535-7163
COUNTRY: United States

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

AB Melanoma is highly resistant to conventional chemotherapy. We have demonstrated that redox regulation in melanoma cells is aberrant, and redox-modulating agents can induce cell apoptosis. We have currently explored the effect of disulfiram (DSF), a member of the dithiocarbamate family, on apoptosis of melanoma cells in vitro. Human metastatic melanoma cells c81-46A, c81-61, and c83-2C were treated with DSF and apoptosis measured. DSF, at a dose of 25-50 ng/ml, consistently caused a 4-6-fold increase in apoptosis. The same dose of DSF did not significantly affect apoptosis in melanocytes. Coincubation of N-acetyl-cysteine reversed the DSF-induced apoptosis. Buthionine sulfoximine (BSO), an inhibitor of gamma-glutamyl-cysteine synthetase, as a single agent caused a approximately 2-fold increase in apoptosis when incubated with melanoma cells for 4 days. BSO slightly enhanced the level of apoptosis induced by DSF (4-10% higher than DSF alone). Intracellular glutathione was remarkably depleted with BSO treatment. DSF did not cause glutathione depletion; however, the ratio of reduced and oxidized glutathione was significantly decreased (14% of control), and N-acetyl-cysteine partially restored the ratio to 30% of control. There was a transient (2-fold) elevation of intracellular superoxide level after 24 h of DSF treatment (before the overt apoptosis). The intracellular H2O2 level progressively decreased with time. DSF decreased the mitochondrial membrane polarization in a time-dependent manner, and there was a significant inverse correlation between apoptosis and mitochondrial membrane polarization. We propose that DSF-induced apoptosis is redox related but involves a different mechanism from BSO-induced apoptosis in tumor cells. Our findings have provided new data for additional understanding of drug-induced apoptosis in melanoma cells and suggests an alternative therapeutic approach to melanoma.

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ACCESSION NUMBER: 0011920175 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: The impact of autologous stem cell transplantation on the prognosis of mantle cell lymphoma: a joint analysis of two prospective studies with 46 patients..
AUTHOR: Dreger, P. (correspondence); Martin, S.; Kuse, R.; Sonnen, R.; Glass, B.; Kroger, N.; Parwaresch, R.; Kneba, M.; Schmitz, N.; Haas, R.
CORPORATE SOURCE: Second Department of Medicine, University of Kiel, Germany.
.
SOURCE: The hematology journal : the official journal of the European Haematology Association / EHA, (2000)
Vol. 1, No. 2, pp. 87-94.
ISSN: 1466-4860
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

AB INTRODUCTION: The purpose of this analysis was to investigate if early sequential high-dose therapy with autologous stem cell transplantation (ASCT) can improve the poor prognosis of patients with disseminated mantle cell lymphoma (MCL). PATIENTS AND METHODS: A joint analysis of two

parallel single center studies was performed. Both were characterized by a sequential high-dose therapy consisting of an intensive chemotherapy ('HAM' or 'Dexa-BEAM') for mobilization of peripheral blood stem cells and induction of minimal disease followed by a total body irradiation-containing myeloablative regimen and ASCT. Forty-six patients with reference panel-confirmed stage III/IV MCL were included. Thirty-four patients were accrued to the protocol immediately after diagnosis ('upfront ASCT' group). These 34 patients received a standard first-line regimen prior to mobilization. The remaining 12 patients were put on the protocol later during the course of their disease ('delayed ASCT' group). RESULTS: All patients were in remission after mobilization chemotherapy and proceeded to ASCT; there were no exclusions due to poor response, poor mobilization, or patient refusal. With a follow-up of 24 (2-73) months post transplant, the event-free and overall survival probabilities at 2 years were 77 and 100% for the upfront ASCT group compared to 30% (P=0.0007) and 54% (P=0.0016) for the delayed ASCT group. Event-free and overall survival tended to be longer in the upfront ASCT group than in the delayed ASCT group also if calculated from initial diagnosis (76 and 93% vs 42 and 63%, respectively, at 4 years after diagnosis; median follow-up 35 months), although this was not statistically significant. Besides timing of ASCT, only spleen size was identified as an independent predictor of survival by univariate and multivariate analysis. CONCLUSION: ASCT is not curative but may improve the prognosis of patients with MCL if performed as part of an intensive first-line treatment strategy. In contrast, the benefits of this approach for salvaging individuals with relapsed disease appear to be limited.

L38 ANSWER 17 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:475749 BIOSIS
DOCUMENT NUMBER: PREV200300475749
TITLE: Enhanced antimelanoma activity after exposure to BSO in combination with disulfiram.
AUTHOR(S): Torres, Carina [Reprint Author]; Fruehauf, John P. [Reprint Author]; Huynh, Lan [Reprint Author]; Parker, Ricardo [Reprint Author]
CORPORATE SOURCE: Oncotech, Inc., Tustin, CA, USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (July 2003) Vol. 44, pp. 923-924. print.
Meeting Info.: 94th Annual Meeting of the American Association for Cancer Research. Washington, DC, USA. July 11-14, 2003.
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Oct 2003
Last Updated on STN: 15 Oct 2003

L38 ANSWER 18 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:337231 BIOSIS
DOCUMENT NUMBER: PREV200300337231
TITLE: Value of Autologous Stem Cell Transplantation in First Line Therapy of Primary CNS Lymphoma.
AUTHOR(S): Colombat, Philippe [Reprint Author]; Mevel, A. Le; Delwail, V.; Foussard, Ch; Brion, A.; Berthou, C.; Bay, J. O.; Quesnel, B.; Quittet, Ph; Himberlin, Ch; Delepine, R.; Desablens, B.
CORPORATE SOURCE: Hopital Bretonneau, Tours, France
SOURCE: Blood, (November 16 2002) Vol. 100, No. 11, pp.

Abstract No. 2533. print.
Meeting Info.: 44th Annual Meeting of the American Society of Hematology. Philadelphia, PA, USA. December 06-10, 2002. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jul 2003
Last Updated on STN: 23 Jul 2003

AB With conventional therapies, ie chemotherapy + radiation therapies, the prognosis of primary CNS lymphoma remain poor. High dose therapy (HD) with autologous stem cell transplantation (ASCT) has given encouraging results as salvage treatment. So, we conducted a phase II study between july 99 and november 2001 evaluating the efficacy of HDT in the first-line treatment of primary CNS lymphoma in patients ltoreq60 years. Patients received initially 2 courses of MVBP (Methotrexate 3 g/m2 on days (D) 1 and 5), VP16 100 mg/m2 on D2, BCNU 100 mg/m2 on D 3, methylprednisolone 60 mg/m2/day on D 1-5) + intrathecal prophylaxis ; in patients in complete or partial remission, peripheral blood stem cells were collected after ifosfamide (1,5 g/m2 on D 1-3) and cytarabine (2 g/m2/day on D 1-2 ; conditioning regimen was BEAM (BCNU 300 mg/m2 on D1), VP16 (400 mg/m2/day on D 2-5), cytarabine (200 mg/m2 on D 2-5) and melphalan (140 mg/m2 on D6 ; after transplantation, patients were irradiated (30 Ggamma in whole brain). Twenty five patients were included in the study. The median age was 51 years (range : 21-60) ; all had diffuse large cell lymphoma ; there were 9 males and 16 females ; ECOG status was 0 in 3 patients (pts), 1 in 10 pts, 2 in 4 pts, 3 in 6 pts and 4 in 2 pts. Twelve patients had one localization and 13 had more than one. Serum LDH level was increased in 6 pts. HDT with ASCT was performed in 16 pts (4 progressions, 3 toxicities and 2 refusals). Out of the 16 pts treated with ASCT, 2 died (1 toxic death and one progression). The overall survival (os) for pts who received ASCT was 82 % at the median follow-up of 18 months and 66 % for the 25 pts. If these first results appear encouraging, a longer follow-up is needed.

L38 ANSWER 19 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:306437 BIOSIS
DOCUMENT NUMBER: PREV200200306437
TITLE: Disulfiram induces apoptosis in human melanoma cells: A redox-related process.
AUTHOR(S): Cen, Dazhi; Gonzalez, Rachel I.; Buckmeier, Julie A.; Kahlon, Ravi S.; Tohidian, Nilou B.; Meyskens, Frank L., Jr. [Reprint author]
CORPORATE SOURCE: Chao Family Comprehensive Cancer Center, College of Medicine, University of California, Irvine, 101 The City Drive South, Building 23, Suite 403, Orange, CA, 92868, USA FLMeyske@uci.edu
SOURCE: Molecular Cancer Therapeutics, (January, 2002) Vol. 1, No. 3, pp. 197-204. print.
ISSN: 1535-7163.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 May 2002
Last Updated on STN: 22 May 2002

AB Melanoma is highly resistant to conventional chemotherapy. We have demonstrated that redox regulation in melanoma cells is aberrant, and redox-modulating agents can induce cell apoptosis. We have currently explored the effect of disulfiram (DSF), a member of the dithiocarbamate family, on apoptosis of melanoma cells in vitro. Human metastatic melanoma cells c81-46A, c81-61, and c83-2C were treated with

DSF and apoptosis measured. DSF, at a dose of 25-50 ng/ml, consistently caused a 4-6-fold increase in apoptosis. The same dose of DSF did not significantly affect apoptosis in melanocytes. Coincubation of N-acetyl-cysteine reversed the DSF-induced apoptosis. Buthionine sulfoximine (BSO), an inhibitor of gamma-glutamyl-cysteine synthetase, as a single agent caused a apprx2-fold increase in apoptosis when incubated with melanoma cells for 4 days. BSO slightly enhanced the level of apoptosis induced by DSF (4-10% higher than DSF alone). Intracellular glutathione was remarkably depleted with BSO treatment. DSF did not cause glutathione depletion; however, the ratio of reduced and oxidized glutathione was significantly decreased (14% of control), and N-acetyl-cysteine partially restored the ratio to 30% of control. There was a transient (2-fold) elevation of intracellular superoxide level after 24 h of DSF treatment (before the overt apoptosis). The intracellular H2O2 level progressively decreased with time. DSF decreased the mitochondrial membrane polarization in a time-dependent manner, and there was a significant inverse correlation between apoptosis and mitochondrial membrane polarization. We propose that DSF-induced apoptosis is redox related but involves a different mechanism from BSO-induced apoptosis in tumor cells. Our findings have provided new data for additional understanding of drug-induced apoptosis in melanoma cells and suggests an alternative therapeutic approach to melanoma.

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ACCESSION NUMBER: 1997:120203 BIOSIS
DOCUMENT NUMBER: PREV199799426706
TITLE: Intensified and high-dose chemotherapy with granulocyte colony-stimulating factor and autologous stem-cell transplantation support as first-line therapy in high-risk diffuse large-cell lymphoma.
AUTHOR(S): Vitolo, Umberto [Reprint author]; Cortellazzo, Segio; Liberati, Anna Maria; Freilone, Roberto; Falda, Michele; Bertini, Marilena; Botto, Barbara; Cinieri, Saverio; Levis, Alessandro; Locatelli, Franco; Lovisone, Elisabetta; Marmont, Filippo; Pizzuti, Michele; Rossi, Andrea; Viero, Piera; Barbui, Tiziano; Grignani, Fausto; Resegotti, Luigi
CORPORATE SOURCE: Div. Ematol., Azienda Ospedaliera S. Giovanni Battista sede Molinette, Corso Bramante 90, 10126 Torino, Italy
SOURCE: Journal of Clinical Oncology, (1997) Vol. 15, No. 2, pp. 491-498.
CODEN: JCONDN. ISSN: 0732-183X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Mar 1997
Last Updated on STN: 25 Mar 1997

AB Purpose: In our previous study with MACOPB, we identified a high-risk group of patients with a poor 3-year survival rate of 29%. These patients were defined as having at diagnosis advanced-stage disease with high tumor burden (TB) and elevated lactate dehydrogenase (LDH) level or bone marrow (BM) involvement. A novel therapeutic scheme was investigated to improve the outcome of these patients. Patients and Methods: Fifty patients with high-risk diffuse large-cell lymphoma (DLCL) were enrolled. The therapeutic scheme includes three phases: induction with 8 weeks of MACOPB; intensification with a 3-day course of mitoxantrone 8 mg/m-2 plus high-dose cytarabine (HDARA-C) 2 g/m-2 every 12 hours plus dexamethasone 4 mg/m-2 every 12 hours (MAD protocol) and granulocyte colony-stimulating factor (G-CSF) 5 mu-g/kg on days 4 to 17 to harvest peripheral-blood progenitor cells (PBPC); consolidation with carmustine (BCNU), etoposide, ARAC, and melphalan (BEAM) regimen; plus autologous stem-cell transplantation (ASCT) with PBPC,

marrow, or both. Results: Thirty-six patients (72%) achieved a complete response (CR), 11 (22%) showed no response (NR), and three (6%) died of toxicity. Among the 22 PRs or NRs after the induction phase, 56% of patients achieved a CR with subsequent intensified therapy. With a median follow-up duration of 32 months, the overall survival and failure-free survival rates were 56% and 50%, respectively. The disease-free survival rate is 69% at 32 months. Leukapheresis after MAD and G-CSF yielded a median of 32 times 10-6/kg CD34+ cells and 80 times 10-4/kg granulocyte-macrophage colony-forming units (CFUGM). Thirty-nine patients were autografted and 11 did not undergo ASCT: six because of disease progression, four due to toxicity, and one because of patient refusal. The median times to achieve engraftment were 11 days (range, 7 to 19) to a neutrophil count greater than 0.5 times 10-9/L and 12 days (range, 8 to 60) to a platelet count greater than 50 times 10-9/L. Conclusion: This sequential scheme with intensified and high-dose chemotherapy with ASCT is feasible with moderate toxicity and may improve the outcome in high-risk DLCL.

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ACCESSION NUMBER: 1989:474876 BIOSIS
DOCUMENT NUMBER: PREV198988110636; BA88:110636
TITLE: MODIFICATION OF CYCLOPHOSPHAMIDE-INDUCED UROTOXICITY BY BUTHIONINE SULFOXIMINE AND DISULFIRAM IN MICE.
AUTHOR(S): ISHIKAWA M [Reprint author]; TAKAYANAGI Y; SASAKI K-I
CORPORATE SOURCE: DEP PHARMACOL TOXICOL, CANCER RES INST, TOHOKU COLL PHARM, 4-4-1 KOMATSUSHIMA, SENDAI 980, JPN
SOURCE: Research Communications in Chemical Pathology and Pharmacology, (1989) Vol. 65, No. 2, pp. 265-268. CODEN: RCOCB8. ISSN: 0034-5164.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 17 Oct 1989
Last Updated on STN: 23 Oct 1989

AB The effect of buthionine sulfoximine (BSO) and disulfiram (DSF) on the urotoxicity induced by cyclosphosphamide (CPA) was examined in mice. Pretreatment of mice with BSO (500 mg/kg, i.p.) 5 hr prior to CPA resulted in enhanced urotoxicity of CPA. In contrast, simultaneous administration of DSF (200 mg/kg, p.o.) decreased the urotoxicity of CPA.

L38 ANSWER 22 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 1985:427341 BIOSIS
DOCUMENT NUMBER: PREV198580097333; BA80:97333
TITLE: HYDROGEN PEROXIDE FROM CELLULAR METABOLISM OF CYSTINE A REQUIREMENT FOR LYSIS OF MURINE TUMOR CELLS BY VERNOLEPIN A GLUTATHIONE-DEPLETING ANTINEOPLASTIC.
AUTHOR(S): ARRICK B A [Reprint author]; GRIFFO W; COHN Z; NATHAN C
CORPORATE SOURCE: ROCKEFELLER UNIV, NEW YORK 10021, USA
SOURCE: Journal of Clinical Investigation, (1985) Vol. 76, No. 2, pp. 567-574. CODEN: JCINAO. ISSN: 0021-9738.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB The sesquiterpene lactone antineoplastic vernolepin acutely depletes murine tumor cell glutathione (GSH), and lyses the cells by an unknown mechanism that is enhanced synergistically by inhibition of GSH synthesis with buthionine sulfoximine. Lysis of P815 mastocytoma cells by vernolepin, with or without BSO, required cystine in the culture

medium. Addition of catalase markedly suppressed vernolepin-mediated cytolysis in cystine-containing media, suggesting the involvement of H_2O_2 in the cytolytic action of vernolepin. Consistent with this, inhibition of tumor cell glutathione disulfide reductase with 1, 3-bis(2-chloroethyl)-1-nitrosourea or inhibition of endogenous catalase with aminotriazole synergistically augmented cytolysis by vernolepin. H_2O_2 was released by suspensions of P815 cells in cystine-containing buffer (63 pmol/10^6 cells · h). Ommission of cystine reduced the rate of H_2O_2 accumulation 10-fold. No H_2O_2 was detected without cells. Cytolysis by vernolepin could be restored in cystine-deficient medium by several other disulfides, themselves non-cytolytic, such as disulfiram and oxidized captopril, as well as by cysteine. Withholding 2 other essential amino acids (leucine or tryptophan) or adding cycloheximide did not interfere with cytolysis by vernolepin. Cellular uptake of disulfides of physiologic and pharmacologic interest may be followed by their intracellular reduction and autooxidation with generation of H_2O_2. This previously unrecognized source of intracellular oxidant stress may be an important component of injury to GSH-depleted cells.

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=> d his

     (FILE 'HOME' ENTERED AT 11:19:38 ON 10 MAY 2010)

     FILE 'CAPLUS' ENTERED AT 11:19:51 ON 10 MAY 2010
L1         2300 S DISULFIRAM
                S DISULFIRAM/CN

     FILE 'REGISTRY' ENTERED AT 11:20:06 ON 10 MAY 2010
L2            1 S DISULFIRAM/CN

     FILE 'CAPLUS' ENTERED AT 11:20:06 ON 10 MAY 2010
L3         3380 S L2
                S DISULFRAM/CN

     FILE 'REGISTRY' ENTERED AT 11:20:23 ON 10 MAY 2010
L4            0 S DISULFRAM/CN

     FILE 'CAPLUS' ENTERED AT 11:20:23 ON 10 MAY 2010
L5            0 S L4
L6         6756 S CURCUMIN

     FILE 'REGISTRY' ENTERED AT 11:20:42 ON 10 MAY 2010
L7            1 S DISULFIRAM/CN
L8            1 S CURCUMIN/CN
L9            1 S BSO/CN
L10           1 S BCNU/CN

     FILE 'CAPLUS' ENTERED AT 11:21:30 ON 10 MAY 2010
L11        3380 S L7
L12        5428 S L8
L13        1947 S L9
L14        3851 S L10
L15     1004074 S CANCER OR TUMOR OR NEOPLASM
L16         224 S L11 AND L15
L17        1654 S L12 AND L15
L18           3 S L13 AND L15
L19        2789 S L14 AND L15
L20          34 S (L16 OR L17) AND (L18 OR L19)
L21          34 DUP REM L20 (0 DUPLICATES REMOVED)
L22          34 S L21
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L23          10 S L21 AND AD<20030718

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:24:17 ON 10 MAY 2010

     FILE 'REGISTRY' ENTERED AT 11:24:40 ON 10 MAY 2010
                SET SMARTSELECT ON
L24          SEL L7 1- CHEM :       72 TERMS
                SET SMARTSELECT OFF

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:24:41 ON 10 MAY 2010
L25       53652 S L24

     FILE 'REGISTRY' ENTERED AT 11:25:20 ON 10 MAY 2010
                SET SMARTSELECT ON
L26          SEL L8 1- CHEM :       42 TERMS
                SET SMARTSELECT OFF

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:25:20 ON 10 MAY 2010
L27       18699 S L26

     FILE 'REGISTRY' ENTERED AT 11:25:33 ON 10 MAY 2010
                SET SMARTSELECT ON
L28          SEL L9 1- CHEM :       13 TERMS
                SET SMARTSELECT OFF

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:25:33 ON 10 MAY 2010
L29        5753 S L28

     FILE 'REGISTRY' ENTERED AT 11:25:41 ON 10 MAY 2010
                SET SMARTSELECT ON
L30          SEL L10 1- CHEM :      21 TERMS
                SET SMARTSELECT OFF

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:25:41 ON 10 MAY 2010
L31       25542 S L30
L32     6349459 S CANCER OR TUMOR OR NEOPLASM
L33        5417 S L25 AND L32
L34        6435 S L27 AND L32
L35        2099 S L29 AND L32
L36       18174 S L31 AND L32
L37          66 S (L33 OR L34) AND (L35 OR L36)
L38          22 S L37 AND PD<20030718

=>

---Logging off of STN---


=>
Executing the logoff script...


=> LOG Y

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    70.12     237.35

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00      -8.50
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STN INTERNATIONAL LOGOFF AT 11:30:13 ON 10 MAY 2010